

=> d his

(FILE 'HOME' ENTERED AT 15:02:41 ON 31 JUL 2003)

FILE 'REGISTRY' ENTERED AT 15:02:51 ON 31 JUL 2003  
E ESTRADIOL/CN  
E 17 ESTRADIOL/CN

FILE 'CAPLUS' ENTERED AT 15:06:31 ON 31 JUL 2003

FILE 'REGISTRY' ENTERED AT 15:06:44 ON 31 JUL 2003  
E 17 ESTRADIOL/CN

FILE 'CAPLUS' ENTERED AT 15:06:44 ON 31 JUL 2003

FILE 'REGISTRY' ENTERED AT 15:07:03 ON 31 JUL 2003  
E ESTRADIOL/CN

L1 1 S E3

FILE 'REGISTRY' ENTERED AT 15:08:17 ON 31 JUL 2003

L2 1 S 50-28-2/RN  
SET NOTICE 1 DISPLAY  
SET NOTICE LOGIN DISPLAY

FILE 'REGISTRY' ENTERED AT 15:08:41 ON 31 JUL 2003  
SET TERMSET E#

DEL SEL Y  
SEL L2 1 RN  
L3 1 S E1/RN  
SET TERMSET LOGIN

FILE 'USPATFULL' ENTERED AT 15:08:44 ON 31 JUL 2003

L4 998 S L3  
L5 30 S L4 AND PY<=1999 AND (HYPERCHOLEST? OR HYPERLIPID? OR LDL OR H  
L6 27 S L5 AND ESTRADIOL

FILE 'REGISTRY' ENTERED AT 15:17:13 ON 31 JUL 2003

SET TERMSET E#  
DEL SEL Y  
SEL L2 1 RN  
L7 1 S E1/RN  
SET TERMSET LOGIN

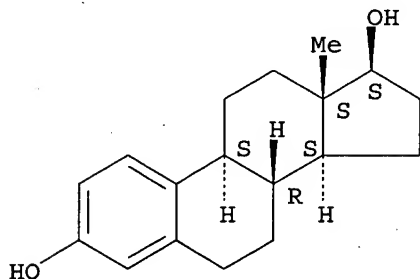
FILE 'CAPLUS' ENTERED AT 15:17:19 ON 31 JUL 2003

L8 49282 S L7  
L9 384 S L8 AND PY<=1999 AND (HYPERCHOLEST? OR HYPERLIPID? OR LDL OR H  
L10 376 S L9 AND ESTRADIOL

=> d rn str cn

L1 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS on STN  
RN 50-28-2 REGISTRY

Absolute stereochemistry.



\*\*PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT\*\*

CN Estra-1,3,5(10)-triene-3,17-diol (17.β)- (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN Estradiol (8CI)

OTHER NAMES:

CN (+)-3,17.β.-Estradiol

CN .β.-Estradiol

CN 13.β.-Methyl-1,3,5(10)-gonatriene-3,17.β.-ol

CN 17.β.-Estradiol

CN 17.β.-Oestradiol

CN 3,17-Epidihydroxyestratriene

CN 3,17.β.-Dihydroxyestra-1,3,5(10)-triene

CN 3,17.β.-Estradiol

CN Aerodiol

CN Altrad

CN Aquadiol

CN Bardiol

CN Beta-estradiol

CN Climaderm

CN Climara

CN Compudose

CN Compudose 200

CN Compudose 365

CN Corpagen

CN Dermestril

CN Dihydrofollicular hormone

CN Dihydrofolliculin

CN Dihydromenformon

CN Dihydrotheelin

CN Dihydroxyestrin

CN Dimenformon

CN Diogyn

CN Diogynets

CN Divigel

CN E 2

CN Encore

CN Epiestriol 50

CN Estra-1,3,5(10)-triene-3,17-diol, (17.β)-

CN Estra-1,3,5(10)-triene-3,17.β.-diol

CN Estrace

CN Estraderm

CN Estraderm TTS

CN Estraderm TTS 100

11, 13,  
25

CN Estraderm TTS 50  
CN Estradot  
CN Estraldine  
CN Estring Vaginal Ring  
CN Estroclim  
CN Estroclim 50  
CN Estrogel  
CN Estrovite  
CN Evorel  
CN Femestral  
CN Femogen

ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for  
DISPLAY

=> d his

(FILE 'HOME' ENTERED AT 11:55:45 ON 31 JUL 2003)

FILE 'EUROPATFULL, PATDPAFULL, PCTFULL, RDISCLOSURE, USPATFULL, USPAT2, WPIDS' ENTERED AT 11:59:20 ON 31 JUL 2003

L1	8332 S	ESTROGEN(S) (ANTIESTROGEN OR (ESTROGEN(3A)RECEPTOR))
L2	478 S	L1(S) (DHEA OR DEHYDROEPIANDROSTERONE OR BISPHOSPHONATE#)
L3	106 S	L1(S) (DHEA OR DEHYDROEPIANDROSTERONE)
L4	25 S	L3 NOT PY>=1999
L5	7 S	L3(S) (CHOLESTEROL OR LDL OR HYPERLIPID? OR HYPERCHOLESTER?)
L6	2 S	L5 NOT PY>=2000

L6 ANSWER 2 OF 2 PCTFULL COPYRIGHT 2003 Univentio on STN  
 ACCESSION NUMBER: 1999063974 PCTFULL ED 20020515  
 TITLE (ENGLISH): MEDICAL USES OF A SELECTIVE ESTROGEN RECEPTOR MODULATOR  
 IN COMBINATION WITH SEX STEROID PRECURSORS  
 TITLE (FRENCH): UTILISATIONS MEDICALES D'UN MODULATEUR DE RECEPTEUR  
 D'OESTROGENES SELECTIF EN ASSOCIATION AVEC DES  
 PRECURSEURS DE STEROIDES SEXUELS  
 INVENTOR(S): LABRIE, Fernand  
 PATENT ASSIGNEE(S): ENDORECHERCHE, INC.  
 LANGUAGE OF PUBL.: English  
 DOCUMENT TYPE: Patent  
 PATENT INFORMATION:

NUMBER	KIND	DATE
WO 9963974	A2	19991216

DESIGNATED STATES

W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK  
 EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP  
 KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL  
 PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU  
 ZA ZW GH GM KE LS MW SD SL SZ UG ZW AM AZ BY KG KZ MD  
 RU TJ TM AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC  
 NL PT SE BF BJ CF CG CI CM GA GN GW ML MR NE SN TD TG

APPLICATION INFO.: WO 1999-CA538 A 19990610  
 PRIORITY INFO.: US 1998-09/096,284 19980611

ABEN Novel methods for the medical treatment and/or inhibition of the  
 development of osteoporosis,  
 breast cancer, **hypercholesterolemia**, **hyperlipidemia**  
 or atherosclerosis in susceptible warm-blooded  
 animals including humans involving administration of selective  
**estrogen receptor** modulator  
 particularly compounds having general structure (I) and an amount of a  
 sex steroid precursor  
 selected from the group consisting of **dehydroepiandrosterone**,  
**dehydroepiandrosterone** sulfate,  
 androst-5-ene-3 $\beta$ ,17 $\beta$ -diol and compounds converted i(in vivo)  
 to one of the foregoing  
 precursor. Further administration of bisphosphonates in combination with  
 selective **estrogen receptor**  
 modulators and/or sex steroid precursor is disclosed for the medical  
 treatment and/or inhibition of  
 the development of osteoporosis. Pharmaceutical compositions. . .

DETD MEDICAL USES OF A SELECTWE **ESTROGEN RECEPTOR**  
 MODULATOR IN  
 COMBINATION WITH SEX STEROID PRECURSORS  
 FIELD OF THE INVENTION

The present invention relates to a method for treating or reducing the  
 likelihood of  
 acquiring osteoporosis, hy  
 percholesterolemia, **hyperlipidemia** or atherosclerosis using a  
 novel combination therapy on susceptible warm-blooded animals, including  
 humans. In  
 particular, the combination includes administering a selective  
**estrogen receptor** modulator  
 (SERNA) and raising the patient's level of precursor to sex steroids,  
 said precursor being  
 selected from the group consisting of **dehydroepiandrosterone**  
 (I)HEA),  
**dehydroepiandrosterone** sulfate (I)FEA-S), and androst  
 ene-3 $\beta$ ,17 $\beta$ -diol (5 $\beta$ diol). The  
 invention also relates to kits and pharmaceutical composition for  
 practicing the foregoing  
 combination.

In another embodiment, the invention provides a method of treating or reducing the risk of acquiring **hypercholesterolemia** comprising increasing levels of a sex steroid precursor selected from the group consisting of **dehydroepiandrosterone**, **dehydroepiandrosterone-sulfate** and androst-4-ene-3 $\beta$ ,17 $\beta$ -diol, in a patient in need of said treatment or said reduction, and further comprising administering to said patient a therapeutically effective amount of a selective **estrogen receptor** modulator as part of a combination therapy.

CLAIMS 1 A method of treating or reducing the risk of acquiring a condition selected from the group consisting of osteoporosis, **hypercholesterolemia**, hyperlipidemia, atherosclerosis, breast cancer, endometrial cancer, uterine cancer, ovarian cancer, vaginal dryness and loss of muscle mass, said method comprising increasing levels of a sex steroid precursor selected from the group consisting of **dehydroepiandrosterone**, **dehydroepiandrosterone-sulfate**, androst-4-ene-3 $\beta$ ,17 $\beta$ -diol and 4-androsten-3,17-dione in a patient in need of said treatment or said reduction, and further comprising administering to said patient a therapeutically effective amount of a selective **estrogen receptor** modulator as part of a combination therapy.

L4 ANSWER 18 OF 25 USPATFULL on STN

ACCESSION NUMBER: 97:94225 USPATFULL  
TITLE: Derivatives of estra 1,3,5(10)triene-17-one, 3-amino compounds and their use  
INVENTOR(S): Li, Pui-Kai, Library, PA, United States  
Selcer, Kyle W., Export, PA, United States  
PATENT ASSIGNEE(S): Duquesne University of the Holy Ghost, Pittsburgh, PA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5677292		19971014
APPLICATION INFO.:	US 1996-607797		19960227 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1994-341410, filed on 17 Nov 1994		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Reamer, James H.		
LEGAL REPRESENTATIVE:	Eckert Seamans Cherin & Mellott		
NUMBER OF CLAIMS:	38		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	6 Drawing Figure(s); 4 Drawing Page(s)		
LINE COUNT:	1007		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DETD The present invention deals with using estra-1,3,5(10)triene-17-one, 3 amino derivatives and the respective **dehydroepiandrosterone** and pregnenolone derivatives as estrone sulfatase inhibitors. Little is known about the metabolism of these compounds and the possible effects. . . metabolite of the estra 1,3,5(10)triene-17-one derivative is estra-1,3,5(10)-triene-17-one, 3-amine (E.sub.1 -NH.sub.2). The procedure and results of the estrogenicity, anti-estrogenicity and **estrogen receptor** binding affinity of E.sub.1 NH.sub.2 are shown below.

L4 ANSWER 19 OF 25 USPATFULL on STN

ACCESSION NUMBER: 97:20519 USPATFULL  
TITLE: Method of treatment of androgen-related diseases  
INVENTOR(S): Labrie, Fernand, Ste-Foy, Canada  
PATENT ASSIGNEE(S): Endorecherche Inc., Canada (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5610150		19970311
APPLICATION INFO.:	US 1995-472512		19950607 (8)
RELATED APPLN. INFO.:	Division of Ser. No. US 1993-98607, filed on 10 Sep 1993 which is a division of Ser. No. US 1992-963278, filed on 19 Oct 1992, now patented, Pat. No. US 5372996 which is a continuation of Ser. No. US 1989-376710, filed on 7 Jul 1989, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Jordan, Charles T.		
ASSISTANT EXAMINER:	Chi, Anthony R.		
LEGAL REPRESENTATIVE:	Ostrolenk, Faber, Gerb & Soffen, LLP		
NUMBER OF CLAIMS:	15		
EXEMPLARY CLAIM:	9		
NUMBER OF DRAWINGS:	1 Drawing Figure(s); 1 Drawing Page(s)		
LINE COUNT:	1639		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

SUMM . . . a schematic representation of the site(s) of action of various drugs, enzymes and hormones. The following abbreviations are used: ER: **estrogen receptor**; AR: **androgen receptor**; DHEA: **dehydroepiandrosterone**; .DELTA..sup.5 -diol:

androst-5-ene-3 .beta., 17.beta.-diol; .DELTA..sup.4 -dione;  
 androstenedione; DHT: dihydrotestosterone; Anti-A: antiandrogen; Anti-E  
**antiestrogen**; ARO: aromatase; 3.beta.-HSD: 3.beta.-  
 hydroxysteroid dehydrogenase, .DELTA..sup.5 -.DELTA..sup.4 isomerase;  
 17.beta.-HSD: 17.beta.-hydroxysteroid dehydrogenase; 1: antiandrogen;  
 17.beta.-hydroxysteroid dehydrogenase activity; 4: **antiestrogen**  
 ; 5; inhibitor of aromatase activity; 6: inhibitor of 3.beta.-HSD  
 activity.

SUMM . . . androgen receptor is shown to stimulate prostatic cancer  
 growth, and is therefore to be prevented. In addition, stimulation of  
 the **estrogen receptor** leads to increased levels of  
 androgen receptors and thus may, in addition, exert direct stimulatory  
 effects on prostatic cancer growth. The action of **estrogens** is  
 therefore to be prevented. Blockers of sex steroid formation from  
**DHEA** and .DELTA..sup.4 -dione in peripheral tissues does not  
 cause inhibition of adrenal glucocorticoid formation. For example,  
 cortisol and aldosterone production. . . result from their inhibition  
 are avoided. The desired inhibition of sex steroid formation is thus  
 aimed selectively at androgens and **estrogens**.

L4 ANSWER 20 OF 25 USPATFULL on STN

ACCESSION NUMBER: 97:5964 USPATFULL  
 TITLE: Combination therapy for prophylaxis and/or treatment of  
 benign prostatic hyperplasia  
 INVENTOR(S): Labrie, Fernand, Ste-Foy, Canada  
 PATENT ASSIGNEE(S): Endorecherche Inc., Quebec, Canada (non-U.S.  
 corporation)

	NUMBER	KIND	DATE
	-----	-----	-----
PATENT INFORMATION:	US 5595985		19970121
APPLICATION INFO.:	US 1993-167450		19931215 (8)
RELATED APPLN. INFO.:	Continuation of Ser. No. US 1992-925883, filed on 6 Aug 1992, now abandoned which is a continuation of Ser. No. US 1989-376700, filed on 7 Jul 1989, now abandoned which is a continuation-in-part of Ser. No. US 1989-322154, filed on 10 Mar 1989, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Kishore, Gollamudi S.		
LEGAL REPRESENTATIVE:	Ostrolenk, Faber, Gerb & Soffen, LLP		
NUMBER OF CLAIMS:	7		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	1 Drawing Figure(s); 1 Drawing Page(s)		
LINE COUNT:	1427		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

DRWD . . . of action of drugs active in the prophylaxis and/or treatment  
 of benign prostatic hyperplasia (BPH). The following abbreviations are  
 used: **DHEA**, **dehydroepiandrosterone**; .DELTA..sup.5  
 -diol, androst-5-ene-3.beta., 17.beta.-diol; .DELTA..sup.4 -dione,  
 androstenedione; T, testosterone; DHT, dihydrotestosterone; E.sub.1,  
 estrone; E.sub.2, 17.beta.-estradiol; ER, **estrogen**  
**receptor**; anti-E, **antiestrogen** (2); anti-A,  
 anti-androgen (5); 17.beta.-HSD, inhibitor of 17.beta.-hydroxysteroid  
 dehydrogenase (4); 3.beta.-HSD, inhibitor of 3.beta.-hydroxysteroid  
 dehydrogenase, .DELTA..sup.5 -.DELTA..sup.4 - isomerase (6),. . .

L10 ANSWER 6 OF 376 CAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 1999:765006 CAPLUS

DOCUMENT NUMBER: 131:346787

TITLE: Double-blind randomized placebo-controlled study of transdermal estrogen replacement therapy on hypertensive postmenopausal women

AUTHOR(S): Modena, Maria Grazia; Molinari, Rosella; Muia, Nicola, Jr.; Castelli, Annadele; Pala, Francesca; Rossi, Rosario

CORPORATE SOURCE: Institute of Cardiology II, Department of Internal Medicine, Policlinico Hospital, University of Modena, Modena, Italy

SOURCE: American Journal of Hypertension (1999), 12(10, Pt. 1), 1000-1008  
CODEN: AJHYE6; ISSN: 0895-7061

PUBLISHER: Elsevier Science Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB We investigated the effects of transdermal 17.beta.-estradiol, combined with std. antihypertensive therapy, on the modification of the cardiovascular risk profile in hypertensive postmenopausal women. In a randomized, double-blind, placebo-controlled study, we enrolled 200 postmenopausal women with mild to moderate hypertension. Patients received 17.beta.-estradiol (50 .mu.g/day, transdermal) and norethisterone acetate (2.5 mg/day, orally) or placebo. At baseline serum total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, glucose, and fibrinogen plasma levels were measured and all subjects underwent complete M-mode and 2-D endocardiograms, which were repeated after 6, 12, and 18 mo of hormonal replacement therapy. Compared with placebo, all values decreased significantly except for HDL cholesterol. In both groups, no modifications were obsd. in echocardiog. parameters, except for left ventricular mean diastolic and systolic wall thickness and left ventricular mass index, which showed a significant decrease in both groups. The redn. was greater in the treated group; the percentage of patients with left ventricular hypertrophy was 46% before randomization and 17.2% after 18 mo of treatment (P <.0001), whereas in group II the percentage was 48% at baseline and 31.5% after 18 mo (P <.05). In conclusion, transdermal 17.beta.-estradiol, assocd. with antihypertensive therapy, may contribute to the redn. of cardiovascular risk profile in hypertensive postmenopausal women.

REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT.

10/052,803

L10 ANSWER 25 OF 376 CAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 1999:341400 CAPLUS

DOCUMENT NUMBER: 131:97765

TITLE: Effect of 17.beta.-**estradiol** in **hypercholesterolemic** rabbits with severe endothelial dysfunction

AUTHOR(S): Do Nascimento, Carlos Antonio; Kauser, Katalin; Rubanyi, Gabor M.

CORPORATE SOURCE: University of Sao Paulo, Sao Paulo, 01246-903, Brazil

SOURCE: American Journal of Physiology (1999), 276(5, Pt. 2), H1788-H1794

CODEN: AJPHAP; ISSN: 0002-9513

PUBLISHER: American Physiological Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB 17.beta.-**Estradiol** prevents early vascular lesion development and may also affect advanced atherosclerosis. To test the antiatherosclerotic effect of estrogen under conditions that resemble more advanced human atherosclerosis with severe endothelial dysfunction, we have investigated the effect of 17.beta.-**estradiol** in **hypercholesterolemic** rabbits treated with the nitric oxide synthase inhibitor N.omega.-nitro-L-arginine Me ester (L-NAME). Chronic L-NAME administration attenuated endothelial nitric oxide (EDNO)-mediated vascular responses leading to significantly accelerated atherosclerotic plaque development. 17.beta.-**Estradiol** treatment alone inhibited aortic lesion formation with concurrent increase in EDNO-mediated responses. The beneficial effect of estrogen persisted in the L-NAME-treated rabbits, suggesting that the antiatherogenic action of 17.beta.-**estradiol** involves NO-independent mechanisms as well. Serum cholesterol levels were not altered by any of the treatments. 17.beta.-**Estradiol** treatment significantly increased EDNO prodn. under these conditions as well. The redn. in plaque size by 17.beta.-**estradiol** was always accompanied by increased EDNO prodn., suggesting a strong assocn. between these two events. The results demonstrate that estrogen treatment may exert protection against atherosclerosis even in patients with severe endothelial dysfunction.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Effect of 17.beta.-**estradiol** in **hypercholesterolemic** rabbits with severe endothelial dysfunction

SO American Journal of Physiology (1999), 276(5, Pt. 2), H1788-H1794

CODEN: AJPHAP; ISSN: 0002-9513

AB 17.beta.-**Estradiol** prevents early vascular lesion development and may also affect advanced atherosclerosis. To test the antiatherosclerotic effect of estrogen under conditions that resemble more advanced human atherosclerosis with severe endothelial dysfunction, we have investigated the effect of 17.beta.-**estradiol** in **hypercholesterolemic** rabbits treated with the nitric oxide synthase inhibitor N.omega.-nitro-L-arginine Me ester (L-NAME). Chronic L-NAME administration attenuated endothelial nitric oxide (EDNO)-mediated vascular responses leading to significantly accelerated atherosclerotic plaque development. 17.beta.-**Estradiol** treatment alone inhibited aortic lesion formation with concurrent increase in EDNO-mediated responses. The beneficial effect of estrogen persisted in the L-NAME-treated rabbits, suggesting that the antiatherogenic action of 17.beta.-**estradiol** involves NO-independent mechanisms as well. Serum cholesterol levels were not altered by any of the treatments. 17.beta.-**Estradiol** treatment significantly increased EDNO prodn. under these conditions as well. The redn. in plaque size by 17.beta.-**estradiol** was always accompanied by increased EDNO prodn., suggesting a strong assocn. between these two events. The results demonstrate that estrogen treatment may exert protection against

atherosclerosis even in patients with severe endothelial dysfunction.

ST **estradiol** endothelium dysfunction **hypercholesterolemia**  
; antiatherosclerotic **estradiol** nitric oxide

IT **Hypercholesterolemia**  
(17.beta.-**estradiol** protection against atherosclerosis in  
**hypercholesterolemic** rabbits with severe endothelial  
dysfunction and nitric oxide role therein)

IT Antiarteriosclerotics  
(antiatherosclerotics; 17.beta.-**estradiol** protection against  
atherosclerosis in **hypercholesterolemic** rabbits with severe  
endothelial dysfunction and nitric oxide role therein)

IT 10102-43-9, Nitric oxide, biological studies  
RL: BAC (Biological activity or effector, except adverse); BPR (Biological  
process); BSU (Biological study, unclassified); MFM (Metabolic formation);  
BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process)  
(17.beta.-**estradiol** protection against atherosclerosis in  
**hypercholesterolemic** rabbits with severe endothelial  
dysfunction and nitric oxide role therein)

IT 50-28-2, 17.beta.-**Estradiol**, biological studies  
RL: BAC (Biological activity or effector, except adverse); BSU (Biological  
study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES  
(Uses)  
(17.beta.-**estradiol** protection against atherosclerosis in  
**hypercholesterolemic** rabbits with severe endothelial  
dysfunction and nitric oxide role therein)

IT 57-88-5, Cholesterol, biological studies  
RL: BOC (Biological occurrence); BSU (Biological study, unclassified);  
BIOL (Biological study); OCCU (Occurrence)  
(17.beta.-**estradiol** protection against atherosclerosis in  
**hypercholesterolemic** rabbits with severe endothelial  
dysfunction and nitric oxide role therein)

IT 57-88-5, Cholest-5-en-3-ol (3.beta.)-, biological studies  
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL  
(Biological study); PROC (Process)  
(blood; 17.beta.-**estradiol** protection against atherosclerosis  
in **hypercholesterolemic** rabbits with severe endothelial  
dysfunction and nitric oxide role therein)